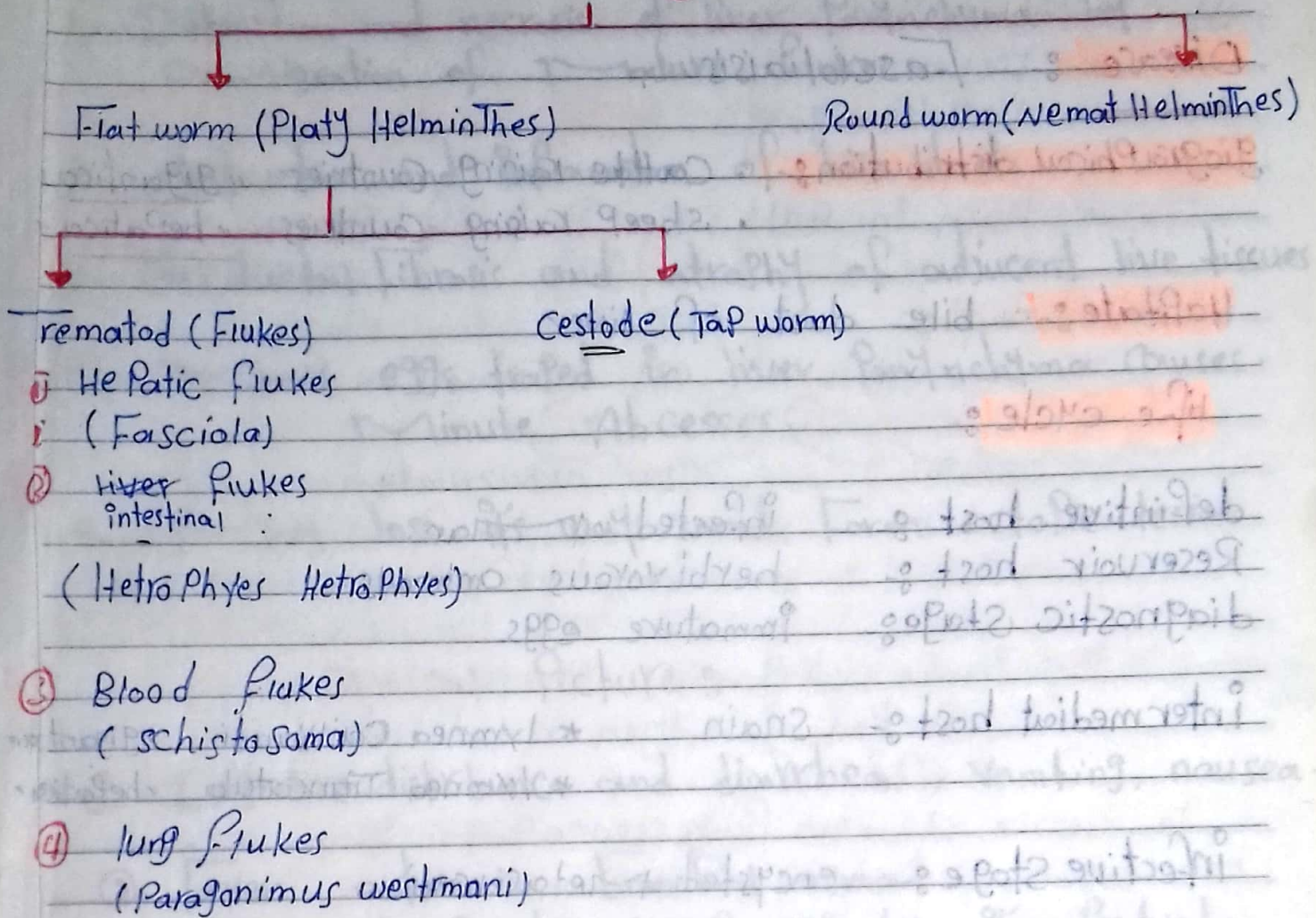


بسم الله الرحمن الرحيم

Helminthology



VISIT...

LANZAROTE
Caliente.COM

Fasciola - liver flukes

① Fasciola gigantica

② Fasciola hepatica

Disease :- Fascioliasis

geographical distribution :-

- cattle raising countries "gigantica"
- sheep raising countries "hepatica"

Habitat :- bile duct of liver.

life cycle :-

definitive host :- infected man.

Reservoir host :- herbivorous animals.

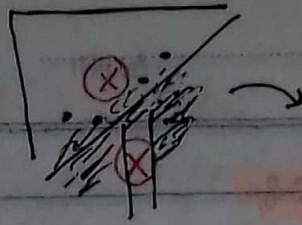
diagnostic stages :- Immature eggs

Intermediate host :- Snail

- * Lymnea Caillaudi "gigantica"
- * Lymnea truncatula "hepatica"

Infective stage :- encysted Metacercaria.

Mode of infection :- ingestion of Raw vegetables or drinking water containing encysted Metacercaria.



Pathogenesis:

- 1- Destruction and necrosis of liver Parenchyma. by migration of mature Flukes.
- 2- obstruction and inflammation of bile duct.
- 3- Peri ductal Fibrosis and atrophy of adjacent live tissues.
- 4- Many eggs trapped in liver Parenchyma causes Minute Abscesses.
- 5- May loss it's way and Form ectopic lesion.

Clinical Picture:

- ① Digestive disturbance and diarrhea, vomiting, nausea.
- ② Fever, urticaria, bronchial asthma.
(Allergic Reaction to Parasite by its Product.)
- ③ Pain in Right upper Part of abdomen.
- ④ Hepatomegaly
- ⑤ jaundice.
- ⑥ cholangitis.
cholecystitis.

1) Diagnosis

1) Clinical

- * Fever, arthralgia
- * Hepato megally
- * Jaundice
- * Pain in Right upper Part of abdomen.

2) Laboratory

- 1) Detection of egg in stool or duodenal aspirate
 - Size $\rightarrow 140 \times 70 \mu$
 - Shape $\rightarrow$ oval, operculated
 - Colour $\rightarrow$ yellowish
 - C $\rightarrow$ Immature ovum.

2) Immuno diagnosis

detection of antigen or antibodies by

- * ELISA
- * Immuno Fluorescence
- * Immuno Electro Phoresis.

3) blood examination

- * Anemia
- * Eosino Philia.

False Fascioliasis

Ingestion of infected raw liver with direct passage of ova in stool.

Diagnosis: Keeping the Patient feeding on liver free diet for one week then Repeat examination.

Absence of egg is sign of false infection.

$\rightarrow$ in false Fascioliasis liver appear healthy.

Treatment:

- * Triclabendazole
- * Dichlorophenol

Prevention and Control:

1. Snail Control
2. Treatment of infected animals.
3. Safe water supply.

Halzoun

Pharyngeal fasciolosis

Condition Result from:

- 1 - ingestion of fresh. Raw sheep or goat liver.
infected with fasciola.
2. linguatula serrata. found in nasal and Respiratory passage of dog.

- Mode of infection:

eating ~~&~~ improperly cooked. infected viscera of
Sheep or goat or Rabbits containing nymphal stage.

Treatment:

- (1) Gargling with Alcoholic drinks.
- (2) Administration of Emetic.
- (3) Tracheostomy.

Control:-

Proper cooking of Animal tissue.

Intestinal Flukes

"Heterophyes heterophyes"

Geographical distribution

Middle East, Far East
Egypt in lakes as Manzala
Brollos, Bardaweel, Karon.

Habitat

Small intestine embedded deeply in villi

Life cycle

- Definitive host → Man.
- Reservoir host → Fish eating animals.
- Diagnostic host → Eggs.
- 1st Intermediate host → Snail (*Pirenella conica*)
- 2nd Intermediate host → Infected Bony, Bony Fish.
- Infective stage → *Leishman cercaria*.
- Mode of infection → Ingestion of improperly cooked or salted fish containing encysted metacercaria.

Disease : Heterophyiasis.

- 1- Irritation of intestinal mucosa with excessive mucous secretion.
- 2- Egg emboli which may reach to vital organ as brain, heart, lung.
- 3- Eosinophilia.

Clinical Picture:

1. Mucous diarrhea (chronic Intermittent)
2. Abdominal Pain (Colic Pain)

Diagnosis:

[1] Clinical diagnosis

- * History of eating grilled or salting bony, bony Fish
- * Clinical Picture.

[2] Parasitological diagnosis:-

- * Detection of egg in stool

Size $30 \times 15 \mu$

Shape oval, Thick shell, operculated, Post. knobs

Colour yellowish brown.

Content mature. Miracidium.

[3] Blood examination:-

- * $\uparrow$ Eosinophilia.

Treatment: Praziquantel

Prevention and Control:-

- * Destruction of snail.
- * Avoid eating Improperly cooked or salting fish.
- * Treatment of the Patient and Reservoir host.

Lung Fluke

"*Paragonimus westermani*"

Disease: Paragonimiasis.

Geographical distribution: Asia, Africa, South America (Far East)

Habitat: cystic cavities in lung.

Life cycle:

Definitive Host: Infected Man.

Reservoir Host: Fish eating animals (shrimps, crabs)

1st intermediate Host: Snail (*Semisulcospira*)

2nd intermediate Host: Shrimps, crabs, Crayfish.

Infective Stage: Microcercous Cercaria.

Mode of infection: Ingestion of improperly cooked shrimps, crabs, crayfish containing encysted metacercaria.

Pathogenesis and Clinical Picture

- ** Acute Inflammation Due to invasion of Fluke to intestinal wall, Peritoneal cavity, Thoracic cavity.
 - Penetration of small intestine: Diarrhea, abdominal pain.
 - Toxic Allergic: Fever, urticaria.
 - Penetration of diaphragm: Cough, dyspnea, chest pain.

** Pulmonary Paragonimiasis

- lung congestion, showing brown nodules containing worm, egg surrounded by fibrous tissue. This leads to chest pain, chronic cough.

→ brown sputum may contain egg.

→ Pleural effusion.

→ Haemoptysis.

→ lung Abscess

→ Emphysema.

* Ectopic lesion

← liver
spleen
brain
eye.

* Diagnosis

① clinical

* History of eating crabs or cray fish.

* Clinical Picture.

② Parasitological diagnosis.

* Detection of egg in sputum or stool.

S 85 x 50 μ

C ~~oval~~ brown

S Thick, oval, operculated

C immature ovum.

* Immuno diagnosis.

For detection of antibodies by.

- ILISA

- IHAAT

- Complement fixation test.

* Blood Examination.

→ Eosinophilia, Anemia.

[2] Radiology diagnosis

- * X-Ray on chest
- * Computerized Tomography. (soft tissues)

Treatment. Praziquantel.

Prevention and Control

- * Destruction of snail
- * Avoid eating improperly cooked crabs.
- * Treatment of Patient and Reservoir host

Schistosoma (blood flukes)

* differ from other Trematod.

1. separate sex.
2. Male. flat and female cylindrical
3. No Redia stage
4. Infective stage Cercaria not encysted meta cercaria.
5. Egg not operculated Has spine.
6. Testes more than two
7. mode of infection skin Penetration.

Species

- Schistoma Haematobium.
- Schistoma mansoni
- Schistoma japonicum.

	S. Haematobium	S. Mansoni	S. japonicum
Disease.	urinary bilharziasis	intestinal bilharziasis	intestinal bilharziasis
Distribution	Africa, middle east Near east, Nile valley	Africa, Saudi Arabia America. Nile Delta.	Far East.
Habitate (definitive host)	Vesical and Pelvic plexus of veins	Inferior mesenteric plexus	sup. & inf mesenteric plexus.
diagnostic host	mature egg	mature egg	mature egg.
Intermediate Stage.	Bulinus truncatus or	Biomphalaria Alexandrina	onchomelania.
Infective Stage.	furca Cercous	Cercaria.	

التاريخ
Penetration of Furco Cercous cercaria aided by?

- 1- Surface tension of drying droplet of water
- 2- Proteolytic Enzyme secreted by Penetration gland.
- 3- lashing movement of tail.

life cycle:

Female in gynaecophoric Canal of the male →
Towards peripheral capillary → Egg → Pass to
urinary system or intestine → Fresh water.
→ Miracidia → [Snail] → Furco Cercous
Cercaria → Penetrates skin → Schistosomula
→ venous circulation → Migration to lung.
→ Heart → systemic circulation. →
hepatic branches of Portal vein → Maturation
→ Migration to Vesical or intestinal Plexus
of vein.

Pathogenesis:

- * Stage of invasion (1-4) days
- * Stage of migration (2-4) weeks
- * Stage of egg deposition and Extrusion (1-2) months.
 - * Stage of egg deposition.
 - * " " Extrusion. [acute stage]
- * Stage of Tissue Reaction (chronic stage).

n.b schistosomula ⇒ cercaria without tail.

→ [1] Stage of Invasion.

local dermatitis, Irritation, Rash. due to cercarial Penetration.

→ [2] Stage of Migration

(a) Metabolic Product Result in toxic and allergic manifestation (Fever, head ch, urticaria, eosinophilia, muscle Pain)

(b) In Lung → - verminous Pneumonitis.
- hemoptysis, Cough.
- minute hemorrhage.

(c) In liver → Enlarged and tender liver.

[3] Stage of Deposition and Extrusion. (acute stage)

In S. Haematobium.	In S. mansoni and Japonicum.
* Terminal haematuria.	* Dysentery with blood
* Frequent micturition.	and mucous in stool.
* Burning Pain.	* Abdominal Pain & Fistula.
	* In S. Japonicum → lead to <u>Katayama Fever</u> .

sheet

Katayama Fever :-

Causes :-
(1) Female lays large number of egg.
(2) High antigen due to Release soluble egg antigen → Stimulate large number of immune system → large number of Antibodies → severe Allergic Reaction.

* Common with S. j and less common with S. H&M

* The Patient suffer from Fever (may last for several week) chills, diarrhea, generalized lymphadenopathy Eosinophilia.

Stage of Tissue Reaction (chronic stage)

Presence of egg in tissue → Stimulate tissue.
 Reaction → Granuloma formation. → AEO
 Attract inflammatory cell → deposition of fibrous
 Tissue → Damage of affected organ
 → loss of its function.

in urinary bilharziasis

- * bladder calcification.
- * ~ fibrosis.
- * Stone formation.
- * Hydro nephrosis.
- * Hydro ureter.
- * Renal failure.
- * Cancer bladder.

in intestinal bilharziasis.

- * Thickening and fibrosis of colon.
- * intestinal polyposis.
- * weight loss.

Ectopic foci.

Egg may swept by blood to reach various organs.
 Causes granuloma formation and fibrosis.

* Embolic lesion in liver.

- 1- Portal hypertension.
- 2- Esophageal varices.
- 3- Hepato spleno megaly.
- 4- Ascites.

* Embolic lesion in lung.

- * Pulmonary hypertension. → Enlargement
 Right side of Heart. → Congestive
 Heart failure. → Cor-Pulmonal.

Diagnosis

Clinical

Laboratory

Radiological
image.

Endoscopy.

1. Clinically

- * History of Contact with infected water.
- * Clinical Picture.

2. Laboratory

(a) detection of egg.

Detection of S.H in urine by

Detection of S.H in stool

→ * sedimentation. method.

* keto Thick Fecal Smear
(to assess intensity of infection)

→ * Examination of last drop of urine passed after 15 minutes of physical Exercise.

* Rectal swab.

→ Test of Viability ;
living egg are translucent and hatch in fresh water
dead egg opaque with dark content, negative hatch in fresh water.

(b) blood examination.

* Anemia due to

- (i) egg Extrusion.
- (ii) Hyper splinism.

* Leukocytosis

* high Eosinophilia.

© Immuno diagnostic test.

- * ILISA ✓
- * IHAT ✓
- * IFAT ✓

[3] Radiological image.

- * X-Ray and ultra Sonography.

[4] Endoscopy.

- * Cystoscopy
- * Colonoscopy
- * Sigmoidoscopy

Detect lesion and
Take biopsy for
search.

Treatment.

urinary bilharziasis

1. Praziquantel
2. Metronidazole.
3. Isoniazid.

intestinal bilharziasis.

- Praziquantel.
- oxamniquine.

(d)

Cercarial dermatitis

Swimmer
itching

Condition that occurs due to Penetration of Cercariae of non human species of Schistoma The skin of man. (Can't go beyond germinal layer)

Clinical Picture

Dermatitis, Itching, Edema, Irritation.
Secondary bacterial infection.

Diagnosis

History of contact of water followed by skin rash.

Treatment:

- Anti Pruritics.
- Anti histaminic.
- Anti biotic.

Acute Fascioliasis

occure as a result of migration of Parasite From The Intestine to and Through the Liver. Causes necrosis of Liver Tissue.

Clinically it is Represented by.

- * Gastro intestinal Problem as nausea, Vomiting, diarrhea.
- * Fever, Rash of skin, Erythema.
- * Hepatomegaly, liver tender.
- Stool examination is negative & during acute Phase.

Chronic Fascioliasis.

occur when Parasite stay in bile duct.

Clinically:-

- * bile duct obstruction and inflammation.
- * Peri ductal fibrosis.
- * cholangitis.
- * cholecystitis.
- * minute abscess around egg.
- * Hepatomegaly
- * obstructive jaundice.

Q: occurrence of terminal hematuria in schistoma.
Hematobium??

Due to deflection of Egg by adult female of S. Hematobium in vesical Plexus of Veins.

Egg Penetrate tissue of urinary bladder to its lumen

Contraction of bladder at the end of micturition lead to Hemorrhage.

Q occurrence of bilharzial or Pulmonal.

Egg of Schistosoma Pass with the blood to Reach to Pulmonary Capillary → Pulmonary Hypertension → Enlargement of Right side of Heart → Congestive Heart Failure → Cor Pulmonal

Q. Occurrence of Anemia in Shistosomiasis?

- Due to 1- Egg extrusion. (iron deficiency anemia)
2- Hypersplenism. (Haemolytic anemia)

General character of cestode.

(6) + (5)

* Flat and segmented (Tap worm).

* live in small intestine.

* Hermaphrodites

* 6 adult cestode live in small intestine of man

* 5 ~ ~ ~ ~ ~ animals.

(extra. intestinal cestode)

* Coracidium. اليرقنة cestode

6. Freezing of fish.

leaf like
 groove
 broad
DiPhyllo bothrium latum

الجين
 Coracidium

disease → Di Phyllo bothriasis.

Geographical distribution: Lake Regions in Europe, America.
(not Present in Egypt).

Habitat: Small intestine of man.

Life cycle?

Definitive host → infected human. (small intestine of man)

Diagnostic stage → Egg in stool.

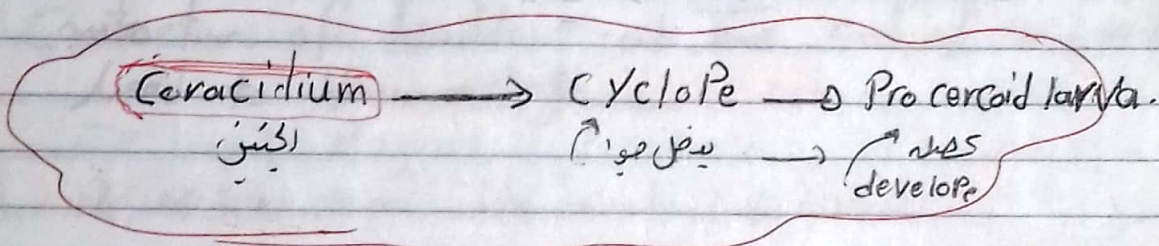
Reservoir host → fish-eating animal.

1st intermediate host → Cyclops. (Coracidium الجين)

2nd intermediate host → Salmon fish.

Infective stage → Plerocercoid larva.

Mode of infection → ingestion of improperly cooked infected salmon, Perch or trout fish.



Cycle:

Adult worm. in. small intestine → Eggs → Faeces
 → Fresh water → Coracidium → 1st intermediate
 Host (Cyclops) → Pro cercoid larva (infective stage)
 → 2nd intermediate host (salmon) → ingestion of
 improperly cooked → Penetration of intestinal
 wall → Small intestine → Maturation

Pathogenesis and Clinical Picture

- 1- Asymptomatic.
- 2- intestinal disturbances
 - colic
 - hunger Pain.
 - nausea.
 - vomiting and diarrhea
 - loss of appetite.
- 3- intestinal obstruction. (in large number).
- 4- neurological manifestation. (caused by absorbed of toxine)
 - Headache.
 - insomnia.
 - Convulsion.
- 5- Pernicious Anemia. Due to (macrocytic hyperchromic)
 - * Toxine of Parasite → ↓ bone marrow
 - * vitamin B₁₂ deficiency. because The Parasite. Compact with The host of Vitamine.

Diagnosis

Clinical

- * Clinical signs, ~~sy~~ Symptoms.
- * dite History.

laboratory

- * detection of egg and segment in faeces.
- * Blood Examination.
Show anemia.

Treatment:

- 1- Praziquantel or
- 2- Niclosamide

+
3- Vitamin B₁₂ given Parenterally.

Prevention and Control

- 1- Mass Treatment
- 2- health education.
- 3- Proper Cooking of fish
- 4- Treatment of infected Patient.
- 5- sanitary disposal of Human excreta
- 6- Freezing of fish.

DiPhyllo bothrium mansonii Proliferum

diseas. g. Sparganosis.

Geographic Distribution: Far Est, Est Africa, U.S.A

Habitat: Small intestine of dog, cat, snakes, Frogs.

* life cycle:

Definitive Host: small intestine of CAT, ~~Frogs~~ Snakes, dog.

Diagnostic Host: only after surgical removal of Larva from infected tissue and identify this Larva.

1st intermediate host: Cyclops.

2nd intermediate host: frogs, snakes, birds, man

Infective stage: Plerocercoid larva.

Mode of infection →

life cycle

Adult worm in small intestine of dog or cat. →

Egg with faeces → fresh water → 1st I.H

→ Pro cercoid larva → 2nd I.H

→ Plerocercoid larva. → Sparganum

→ any Tissues. → Sparganosis.

Q: Why is man blind End of cycle??

because he not eaten by other animals.

Sparganosis

def: Infection of human tissues by plerocercoid larva of *D. manoni* or *D. proliferum*.

Mode of infection:-

- ① ingestion of under cooked or plerocercoid larva of infected Frogs, Snakes, birds.
- ② drinking water containing infected cyclop.
- ③ applying fresh of infected frog, snake, birds as Poultrice to inflamed tissue as eye or skin

Pathogenesis

- depend on the tissue invaded by plerocercoid larva
- 1- Skin → inflammation, edema and urticaria.
 - 2- Eye → Painful edematous, congestive Ptosis.
 - 3- Degenerated larva causes inflammation and necrosis but no fibrosis.
 - 4- Patient may suffer from Eosinophilia, Fever, edema, urticaria.

Diagnosis

* Finding of this larva in Tissue and surgical removal for identification of this Larva.

* Blood Examination → ↑ Eosinophilia.

Treatment :

Surgical Removal
difficult in Sporangium Proliferum. Due to
Proliferation and spread to other Tissue.

Prevention and Control

- ① Filtration or boiled water before drinking.
- ② Proper cooking flesh of frogs, snakes, birds.
- ③ Avoid applying. Flesh of frog, snakes, birds
as poultice to inflamed tissue.

	<u>Taenia saginata</u>	<u>Taenia Solium</u>
Disease	Taeniasis Saginata	Taeniasis Solium.
Geographical Distribution	Cosmopolitan. especially in cattle rising Countrise.	especially in Pig rising. Counterise.
long.	4 - 10 m.	2 - 4 m.
Habitate	Small intestine of man	Small intestine of man.
Definitive host	small intestine of man.	small intestine of man.
Diagnostic host	immature egg. (hexacanth oncospher)	immature egg.
Intermediate host	Cattle	Pig (man) <u>meat</u>
Infective stage	Cysticercus bovis.	Cysticercus Cellulose.
Mode of infection	ingestion of under cooked infected beef	ingestion of under cooked infected Pork.
Life cycle	Adult worm in small intestine → Egg → Grass → ingestion by cattle → Penetrate intestinal wall → blood → muscle → Cysticercus bovis → ingestion by definitive host → small intestine → maturation	Adult worm in small intestine → Egg → Grass → ingestion by Pig → penetrate intestinal wall → muscle → Cysticercus Cellulosis → ingestion by man. → small intestine. → maturation

Taenia Saginata

- 1- intest disturbance
- 2- intestinal obstruction.
- 3- loss of weight and hunger Pain.
- 4- neurological manifestation
- Due to toxin.
- 5- ↑↓ (migrate)

- Appendicitis
- Cholangitis cause

6- Migration of segment creeping out of anus causes irritation, itching and worry.

1* Detection of egg or segment in faeces.

2- Detection of motile segment under the clothes or on bed.

staining of egg by Ziehl Neelsen stain.

stained egg → Taenia saginata egg

Praziquantel

Niclosamide

Taenia Solium

- intestinal disturbance.
- 2- intestinal obstruction.
- 3- loss of weight hunger Pain.
- 4- neurological manifestation
- Due to toxin.

if man. ingest the eggs the larval stage develop in Extra. intestinal Tissue → Cysticercosis

1* Detection of eggs in faeces.

differentiation by Ziehl Neelsen stain.

2- detection of gravid segment in faeces.

unstained egg → Taenia solium egg.

- Praziquantel or Niclosamide

2- Saline Purge is given

1-2 hours later to wash the egg to prevent Cysticercosis.

Pathogenesis
Clinical Picture

Diagnosis

Treatment

Prevention and Control ?

1. Sanitary disposal of human excreta.
2. Treatment of infected man.
3. Health Education.
4. Proper cooking of meat (beef or pork)
5. Proper inspection of slaughtered cattle or Pigs.

Q. what happen when ingestion of *Taenia saginata* egg by human ??

Nothing happens because it dead by gastric juices.

Q. what happen when ingestion of *Taenia solium* by human?

Cysticercous cellulosa → human tissues.

→ Extra intestinal tissue → cysticercosis.

(Cysticercosis)

def: invasion of human tissue by cysticercus cellulosae, the larval stage of *T. solium*.

Mode of infection

1- Ingestion of *T. solium* egg in contaminated Food or drink Hetero infection.

2- External autoinfection.

Hand to mouth infection in Patient having adult worm in intestine.

3- internal auto infection.

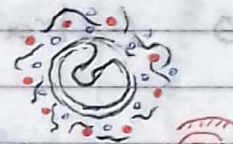
Patient having adult worm in his intestine.

Some segment detached and ascend by anti Peristaltic movement to the stomach.

Then descend again to intestine where egg hatch and causes cysticercosis.

Pathogenesis

and

Clinical Picture

1- sites: brain, subcutaneous tissue, Heart, eye.

2- Cysticercus cellulosae produce inflammatory Reaction. End by Fibrosis and calcification.

3- muscle Pain.

4- Fever and Eosinophilia.

5- subcutaneous cyst easily Palpated (lipoma)

6- eye cyst lead to Visual disturbance

7- cyst affecting nerve lead to neurological disturbance.

Diagnosis

Clinical	Radiological	Laboratory
* History of intestinal infection with <i>T. solium</i>	* X-Ray show calcified cysts as Spindle shape	* Eosinophilia (Blood Examination)
* Biopsy Taken From nodule in skin or muscle	* MRI magnetic resonance	* IHT indirect Hemagglutination test
	* C-T computerized Tomography (soft tissue)	* ELISA
		* Intra dermal Test.

Treatment

1. Surgical Removal
2. Praziquantel.
3. Anti simultaneous administration of steroid.
To Relieve intense of inflammatory Reaction to antigenic material of dead larvae
4. Vit D and calcium to help calcification.
To localize infection.

Prevention and Control.

1. Sanitary disposal of Human Excreta.
2. Proper inspection of ~~st~~ slaughtered cattle or Pigs.
3. Health education
4. Treatment of infected Patient.
5. Proper washing vegetable.

Echinococcus granulosus

Geographical distribution: sheep Raising Countries

disease: hydatid disease

Habitat: Small intestine of dog and canines

but not man (carry adult stage)

(non-fertile host) Small intestine of dog and canines.

Diagnostic host: hydatid cyst.

Intermediate host: Herbivorous animal (carry young stage)

(sheep, cow, camel, man)

not infective stage: Egg.

Mode of infection:

(1) Hand to mouth

(2) Food or drink infected by faeces

(3) of Animal

Life cycle:

Adult in small intestine of dog → egg (oncosphere)

→ faeces → grass → ingestion by intermediate

host → Penetrates small intestine → Pass to blood by

lymphatic or venules, → various part of body.

→ grow slowly. → Hydatid cyst.

Pathogenesis and Clinical Picture.

According to site of cyst :-

In Liver (hepatic cyst) 66 %

Clinical Picture.

1- No symptoms until it expand

2- obstructive jaundice

→ jaundice

3- Rupture: lead to

* Secondary new cysts with hydatid sand of germinal layer seeded in Peritoneal cavity

* open into bile duct

→ intermitted jaundice + Fever + Eosinophilia

* Sudden Rupture of cyst lead to Allergic manifestation Anaphylactic shock.

→ Anaphylactic shock

In Lung (Pulmonary cyst) 22 %

Cyst Present in lung tissue causes

* Transient Thoracic Pain.

* Shortness of breath

* Haemoptysis.

Transform into chronic abscess

* Sudden attack of cough

with sputum contain blood, mucous, hydatid material

in brain. 1 %.

- increases intracranial tension.

lead to

* Headch

* epilepsy.

in Kidney 3 %.

- intermitten haematuria.

* Hydatid sand may Present in urine.

in bone. 2 % (osseous cyst).

In bone no fibrous and laminated layer only
germinal layer Present.

- Growth of osseous cyst cause

* Bone Erosion

* Trabeculae destruction

* Spontaneous fracture.

liver

brain.

lung

bone.

Kidney

Diagnosis.

- ① History of contact with dog and clinical Picture.
(slowly growing cystic Tumor).
- (② X-Ray especially in Pulmonary cyst and calcified cyst.
- ③ Ultra sound and C.T in detection of uncalcified cyst.
- (④ Serological test $\begin{cases} \text{IHA} \\ \text{ILISA} \end{cases}$
- ⑤ blood Examination $\rightarrow$ Eosinophilia.
- (⑥ Molecular diagnosis by P.C.R.
- ⑦ Aspiration cytology
- * ⑧ Intra dermal test (casoni test)
It may give false. Result in 18% of cases.
Now not Preferred due to Allergic Reaction
- * ⑨ Sputum examination $\Rightarrow$ may find fragment of cyst ruptured cyst.

Treatment

- ① Surgical Treatment
- ②PAIR Technique $\begin{cases} \text{Per cutaneous} \\ \text{Aspiration} \\ \text{injection.} \\ \text{Reaspiration} \end{cases}$
- ③ Albendazole, Praziquantel.

E. GranulosusE. MultilocularisM. MulticepsHabitat

Small intestine of dogs

in Small intestine of
Foxes, wolves.Small intestine
of dog.Mode of infection

Ingestion of egg by

Ingestion of egg

Ingestion of eggs by

(1) Hand to mouth from
fur of dogs.during Trapping or
Skinning of infected
foxes or wolves(1) Hand to mouth
from fur of dog.(2) Contaminated Food
or drink with faeces
of infected dog.(2) Contaminated Food
or drinksForm

Hydatid cyst

Alveolar cyst

Coenurus cyst

Favorite site in man

Liver 66%

Liver 90%

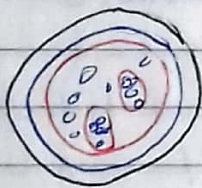
Brain

Hydatid cystAlveolar cystCoenurus cyst

* unilocular cyst

Multi locular cyst

unilocular cyst

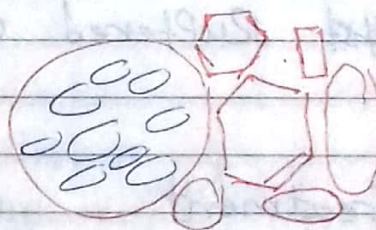


Fibrous

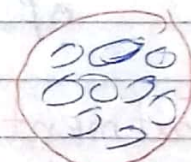
laminated

Germinal

layer



only Germinal layer

only Germinal
layer

Egg < diagnostic
infective stage

Hymenolepis nana

Disease: Hymenolepiasis

Distribution: Found in warm countries, Egypt and Sudan.

Habitat: Small intestine of man. more common in children.

N.B. - Adult live in small intestine.

- Larva live in sub mucosa embedded in villi. So no intermediate host

Man act as
 < definitive host
intermediate host.

Definitive host: Small intestine of man

Diagnostic host: Egg

Intermediate host: no intermediate host

Cysticercoid nana embedded in villi of S.I
or flea larva of Rat

Diagnostic host: Egg in stool.

Infective stage: Egg or cysticercoid larva in Rat flea.

Mode of infection: (1) Auto infection. (hand to mouth)
(2) ingestion of Contaminated food and water

Life cycle:

(A) Direct: Adult in small intestine of man → eggs
→ Faeces → ingestion by man → Penetrat
mucosa of small intestine → cysticercoid larva →
Return to Lumen of small intestine → Adult stage.

Indirect cycle: - Adult in small intestine of man.
— eggs — Faeces — eaten by Rat flea
— cysticercoid larva — ingestion by man.
— Adult stage.

Pathogenesis and clinical Picture.

light infection:	Abdominal Pain.
Produce no symptoms	loss of Aptite.
	Diarrhea, Vomiting.
Heavy infection Produce	absorb Toxic Product of worm lead to
enteritis as a result of	- urticaria
ulceration of mucosa.	- nervous manifestation (dizziness, insomnia, convulsion)

1. Diagnosis: - detection of egg in stool.

Treatment: - Praziquantel

- 2- Treatment should be Repeated after 10 days due to liberation of larva from villi
- 3- Treatment all member of family at same time.

Control:

1. Personal hygien
2. Mass Treatment
3. Avoid infected food and drink
4. Environmental Sanitation

DIPYLIDIUM CANINUM

(dog Tap worm)

disease:- dipylidiasis

Habitat:- Small intestine of man.

Definitive host:- infected human.

Diagnostic host:- Gravid segment or egg capsule in stool.

Intermediate host:- Flea Larva of dog.

Infective stage:- cysticercoid caninum in dog flea.

Mode of infection:- ingestion of infected dog flea in contaminated food or drink.

Life cycle:-

Adult in small intestine of man → egg → fleas
→ eaten by dog flea → cysticercoid larva
→ ingested by man → Adult stage.

Pathogenesis and clinical picture

Light infection:- Produce no symptoms

Heavy infection:- abdominal Pain, Pruritus may occur.
intestinal disturbance.

Diagnosis:- finding of gravid segment or egg capsule in stool.

Treatment:- as H. nana.

Hymenolepis diminuta (Rat Tape worm)

directus: Hymenolepis diminuta.

Habitat: - small intestine of Rat and mice. (Adult)
occasionally Small intestine of man.

Definitive host: - Small intestine of man.

Diagnostic host: - Eggs in stool.

intermediate host: - Flea Larva of Rat.

infective host: - Cystercoid diminuta in Rat flea.

Mode of infection: - ingestion of infected Rat flea in contaminated food or drink.

Life cycle:-

Adult in small intestine → Eggs → Faeces
→ eaten by Rat Flea → Cystercoid Larva
→ ingestion of Rat flea by man →
Sub intestinal mucosa → Adult stage.

Treatment: - as H. nana.

Pathogenesis

Diagnosis and clinical Pictures:-

- 1- light infection: asymptomatic.
- 2- mild intestinal disturbance.

Diagnosis: - detection of egg in stool.

H. nana.

disease. - Hymenolepiasis nana.

Habitat. Small intestine of man.

Definitive H. Small intestine of man.

Diagnostic. Eggs in stool

Intermediate. Cercoid nana embedded in submucosa of man. or Flea larva (Rat)

Infective. egg or cercoid larva of (Rat) flea.

Mode of infection. 1. ingestion of contaminated food or water
2. Hand to mouth infection

D. Caninum.

Dipylidiosis Caninum.

Small intestine of dog
occasionally small intestine of man

Small intestine of man

Egg capsule in stool.

Flea larva of (dog).

Cercoid Caninum in dog flea

ingestion of infected dog flea
in contaminated food or drink

H. diminuta.

Hymenolepiasis diminuta.

Small intestine of Rat.
occasionally small intestine of man

small intestine of man.

Eggs in stool.

Flea larva of (Rat)

Cercoid diminuta in (Rat) flea.

ingestion of infected Rat flea
in contaminated food or drink

Cestod

2D
2T
2H

(5)

Extra intestinal cestod

- 1- Echinococcus granulosus
- 2- Echinococcus Multilocularis
- 3- Multiceps Multiceps.
- 4- Diphylo bothrium mansonii
- 5- Diphylo bothrium Proliferum.

intestinal cestod

- 1- Diphylo bothrium Latum
- 2- Dipylidium Caninum.
- 3- Tenia Saginata.
- 4- Taenia Solium.
- 5- Hymenolepis nana.
- 6- Hymenolepis diminuta.

1- Echinococcus granulosus

→ Hydatid cyst

2- Echinococcus multilocularis

→ alveolar cyst

3- Multiceps Multiceps

→ Coenurus Cerebralis cyst

4- Diphylo bothrium. mansonii

→ Sparganosis.

5- Diphylo bothrium Proliferum.

+

6- larva of Tenia solium.

→ cysticercosis

الآفة الدورية للدماغ

1- PAIR.

2- Vitamin B₁₂ deficiency in D. bothrium latum.

3- Clinical Type of cysticercosis

4- diagnosis of Sparganosis.

5-

Clinical Type of cysticercosis ??

① neuro cysticercosis :- 60%

Most of Patient with cysticercosis have central nervous system manifestation as → Nausea, seizure, Vomiting, Lethargy, Headach, Confusion, numbness and weakness

② cysticercosis of skeletal muscle and sub cutaneous tissue.

Causing sub cutaneous nodule about 5-1cm in size. Fixed in Portion causing muscle weakness and fatigue.

③ ocular cysticercosis.

either intra ocular that causes vision change. or sub conjunctival cysticercosis.

PAIR

non surgical approach (way) for Treatment of Hydatid cyst.

Percutaneous Aspiration of isom cyst fluid.

injection of cyst with scolosidal solution

Reaspiration of scolosidal solution

Then follow up cyst Regression by using ultra sound

Vitamin B₁₂ deficiency associated with diphylo -
- bothrium latum infection ??

- ① Due to Completion of worm with host for vit B₁₂
- ② impairment Reaction between extrinsic and intrinsic Factor of Castle. → enhance. Pernicious anemia
- ③ inadequate supply of Vitamin B₁₂ in diet.
- ④ Toxine of Parasite causing bone marrow depression.

Coenurosis. (Multiceps Multiceps)

def. human infection with Coenurus cerebralis the larval stage of Multiceps, Multiceps.

Habitat → Adult worm inhabits small intestine of dog.
→ larva inhabits brain and spinal cord of Herbivorous animal.

Morphology → unilocular cyst formed only germinal layer

cycle Adult worm → Eggs → Faeces → Grass
→ herbivorous animal → Small intestine.
Penetrates intestinal wall → brain →
develop into Coenurus larva.

Clinical Picture → lesion in CNS
→ Headach
→ Convulsion
→ epilepsy.

Diagnosis → only after removal of larva and identify it

Treatment → Surgical Removal.

Ascaris lumbricoides

Ascaris - worm.

lumbricoid - earth worm like.

Geographical distribution: Cosmo Politan. (الإصابات موزونة في كل مكان)

Habitat: Man (only man) Lumen of small intestine.

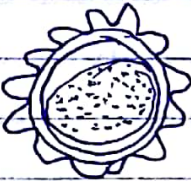
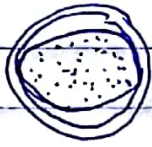
Life cycle.

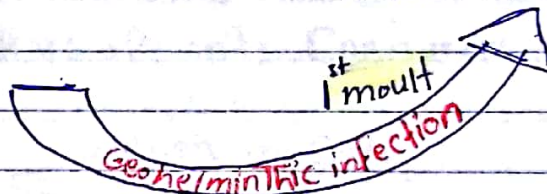
Defensive host: Man

Diagnostic stage: [Ascaris egg] in stool

Infective stage: [embryonated eggs]

Mode of infection: ingestion of embryonated egg in contaminated food.

Diagnostic stage.			Infective stage.
Ascaris egg in stool			Embryonated egg.
			
unfertilized egg	Fertilized egg.	Decorticated egg.	egg contain. 2 nd stage of Rhabditi form. larva.
long	2 layer	Fertilized egg.	
narrow	Smooth	without	
ill-defined	sub-mamillation.	mamillation	Rhabditi form larva develop in egg on ground.
mamillation - immature.			
	- brown.		



Life cycle: Adult worm -> egg -> faeces -> soil -> 1st Rhabditi form larva -> 1st moult -> 2nd Rhabditi form larva -> ingestion -> Penetrate small intestine -> Venous blood -> Rt. side of Heart -> Pulmonary artery -> lung. enter alveoli -> 2nd moult -> 3rd moult -> ascend in. Respiratory tract -> swallowed -> small intestine -> 4th moult -> Adult worm.

Pathogenesis and clinical picture

(1) Migration stage (During larval migration)

** local inflammatory reaction around the larva leading to Pneumonitis. manifested by dry cough & long grade fever & dyspnea & eosinophilia.

** Allergic manifestation as asthma / edema of the lip.

** Some larva may reach to left side of heart and distributed as emboli to various organ.

(2) Adult worm in small intestine

** Digestive disturbance

- Abdominal Pain, Colic
- nausea, vomiting
- Distention, Dyspepsia

Due to Production of lytic enzyme. That interfere with Protein digestion and Fat digestion.
(anti Pepsin, Anti Trypsin)

(3) Complication.

① Traumatic effect - Due to irritation of worm may go to

- 1 * bile duct obstructive jaundice.
- 2 * liver - liver abscesses
- 3 * Appendix - Appendicitis.
- 4 * Ampulla of Vater - Acute hemorrhagic pancreatitis.
- 5 * Peritonium - Peritonitis (Perforation of intestine)
- 6 * May Pass to Stomach - vomiting.
- 7 * May Pass to Trachea - suffocation.

- * May causes Volvulus (Portion of small intestine twists around it self)
- * May causes intussusception (Portion of intestine enter into another Portion)

(b) **Toxic effect**, (caused by toxins of living or dead worm)

Due to hyper sensitivity to the worm antigen. Manifested by fever & urticaria & asthma & edema.
insomnia & nervous irritability & convulsion.

(c) **larva in ectopic site**:

Visceral larva migrans

Diagnosis $\leftarrow$ clinical
laboratory
Radiology

Clinical:

Transient cough & dyspnea which disappear after 1-2 weeks followed by vague (vague) abdominal manifestation.

Laboratory:

- 1- Egg in faeces
- 2- Sputum examination. Finding larva in sputum with blood
- 3- Eosinophilia
- 4- Adult in vomits & faeces.

Radiology:

* X-Ray - lung show scattered mottling called Loeffler's syndrome.

* Barium meal

Treatment

- 1- Albendazole or Mebendazole.
2. in mixed infection. Treat Ascaris infection first
To prevent stimulating worm to unwanted activity.

3. Surgical Treatment

Prevention & Control

1. Mass Treatment
2. washing hand before meals
3. Sanitary disposal of human faeces.
4. Proper washing of fruits and vegetables.
5. Night Soil Should not be used as fertilizer.

Trichuris Trichiura (Long intestine)

disease: Trichuriasis.

Geographical Distribution: Cosmopolitan.

Habitate: Caecum and adjacent part.

Definitive host: Man.

Diagnostic stage: Egg in stool.

Infective stage: Embryonated egg.

Mode of infection: ingestion of contaminated green vegetable with embryonated Trichuris egg.

(ingestion of embryonated egg in contaminated food or drink)

Life cycle:-

Adult in Caecum → egg → soil → Rhabditiform larva
→ ingestion → lower part of small intestine.
Caecum → 4th moults → adult

Pathogenesis and clinical picture

The anterior part of worm embedded in mucosa causing inflammation and irritation of mucosa with hemorrhage. Secondary infection. Result in submucosal abscesses & ulcer.

Mild infection: usually asymptomatic.

Moderate infection:-

- * nausea, vomiting.
- * lower abdominal pain.
- * bloody diarrhea.

Heavy infection:

① Dysentery:-

Anterior part of worm embedded in intestinal mucosa. Cause edematous, hyperaemic, fragile mucosa. Stool with mucus and blood.

② Perforation of intestinal wall → Proctitis.

③ invade Appendix → Appendicitis

④ Anemia. Due to

- blood loss (bleeding) causing Microcytic hypochromic anemia.
- Toxine product of worm causing Macrocytic hyperchromic anemia. (↓ bone marrow)

⑤ Eosinophilia

⑥ Rectal Prolapse. (anal tone is lost due to infection)

Diagnosis:-

- ① Finding egg in stool
- ② Rectal examination by Proctoscope.
- ③ Air Contrast barium enema (علاج)
- ④ blood Examination. * Anemia
* Eosinophilia.

Treatment:-

1. Albendazole Mebendazole.
2. Repeated course is necessary.
3. Give anti diarrhea before Treatment.

4. helminthic causes appendicitis.

- ① Ascaris lumbricoides
- ② Trichuris Trichiura.
- ③ Enterobius vermicularis.
- ④ Taenia Saginata infection.

Common in children:-

* Enterobius

* Ascaris.

Enterobius Vermicularis

Disease: Enterobiasis.

Distribution: Cosmopolitan.

Habitode: Caecum and adjacent Parts

Defensive Host: Man only.

Diagnostic Host: egg [becom infective in few hours]

egg found mainly in Perianal skin.

infective stage: infective egg containing larva.

Mode of infection

Ingestion of infective eggs containing larva through.

* Auto infection. (Hand to mouth)

* Contaminated food or drink.

* Air-borne infection.

* Retro infection.

* Handling Contaminated linen, ^{قماش} clothingPathoLife cycle

Adult —————> Female Migrate towards anal opening.
 lay sticky eggs on Perianal area. —> infection.
 by any mode. —> larvae —> mouth —> adult.

Pathogenesis and Clinical picture:

- 1- Pruritis ani $\rightarrow$ itching in the Perianal area especially at night
- 2- nervous irritability, hyperactivity, insomnia Due to Pruritis ani
- 3- Female $\rightarrow$ migrate to Ectopic site. stimulate Granuloma formation. Through migration to
 - (a) Vagina $\rightarrow$ Vulva Vaginitis also may migrate to uterus and fallopian tube
 - (b) urinary tract $\rightarrow$ urinary tract infection and involuntary micturition (enuresis)
 - (c) Appendix $\rightarrow$ Appendicitis
 - (d) intestine $\rightarrow$ diarrhea & abdominal Pain
 - (e) Peritoneal cavity (Through urine tube) $\rightarrow$ Peritonitis

Diagnosis

Clinically: Pruritis ani at night.

Laboratory:-

- I] Swabbing of Perianal area to detect eggs by.
 - (a) N.I.H. swab (national institute of Health) $\rightarrow$ The Perianal area is swabbed in the morning before defecation or bathing. with cellophan Paper folded and tied to Tip of a glass Rod and inserted in test Tube. $\rightarrow$ The cellophan stretched in slide and examined microscopically.

- ① Scotch adhesive Tap. → sticky side is pressed against Perianal area. Then Put in slide and examined microscopically.
- ② Adult worm may be seen in stool or Perianal area
- ③ egg Rarely found in stool

Treatment

1. Albendazole or Mebendazole it should be repeated after two weeks.
All member of family should be simultaneously treated
2. local application of white oxide of mercury around the anus. (white Precipitate ointment)
To
 - * Relieves itching.
 - * Kill female worm coming out of anus
 - * Prevent dispersal of eggs.

Epidemiology :- (causes of spread)

- (1) Egg become mature and infective in few hour
- (2) Pruritis lead to auto infection.
- (3) Prevalent in Egypt.

Prevention and Control:-

- (1) Mass Treatment
- (2) Personal hygiene
- (3) Food Protection
- (4) infected children should use tight trousers at night
- (5) Toilet seats disinfected frequently.

Ancylostoma Duodenal

like hook

bent

mouth

Disease: Ancylostomiasis.

China, India.

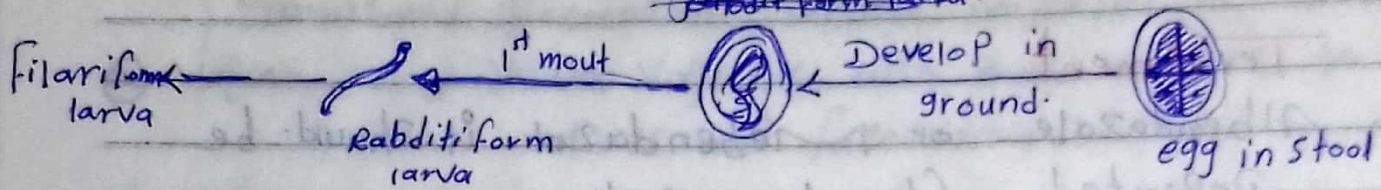
Geographical distribution: Mediterranean, North Africa, South America

Habitat: small intestine (jejunum)

Definitive Host: Man only.

Infective stage: Filariform larva.

~~Rabbit form larva.~~



Mode of infection:

Penetration of Filariform larva The skin of man when he walks bare footed on soil containing larva.

The Filariform larva is

- (1) Positive hydro tropism — attraction towards water
- (2) Thermotropism — attraction towards warmth
- (3) Histotropism — Tissue.
- (4) Negative geotropism — goes up against gravity.

Life cycle:

Adult in Small intestine — eggs — Faeces.

— Soil — 1st stage of Rabbitiform larva — 1st moult

— 2nd stage of Rabbitiform larva — 2nd moult

— infective Filariform larva — Penetrates Skin

— Venules or lymphatics — Rt. side of Heart

— lung — 3rd moult — Penetrates alveoli

— Migrate through Trachea — Swallowed

— Small intestine — 4th moult — adult.

Pathogenesis and clinical picture :-

1] Skin lesion. (invasion stage)

It dermatitis and itching.

Secondary infection at the site of larva lead to formation of Papule, Pustule, Vesicle.

2] Pulmonary lesion. (Migration stage)

Passage of larva in the lung lead to minute hemorrhage & verminous Pneumonitis.

Cough, asthma, Fever, Eosinophilia. [Loeffler's Syndrome]

3] Intestinal lesion (intestinal stage)

a) Hemorrhage Result from attachment of Parasite to the mucos by its cutting teeth. Worm change its site of attachment to mucosa to other site causing minute ulcer.

b) Hypochromic Microcytic anemia Result from chronic blood loss and decrease iron store. → Pallor
Fatigue, dyspnea, Tachycardia

c) Retardation of Physical and mental development.

d) Hypoproteinemia → subcutaneous edema.

e) GIT → nausea, vomiting, diarrhea, Melena

f) Pica → habitual ingestion of non food substance as soil.

Diagnosis.

- 1- Clinical : clinical sign and symptoms.
- 2- laboratory
 - * Detection of Egg in stool.
 - * Determination of Anemia. (Hypochromic microscopies)
 - * Testing for occult blood in stool (الدم الخفي)

Treatment

- 1- Albendazol or Mebendazol (Vermox)
- 2- Iron Supplement (iron therapy)
- 3- Ptn. Rich. diet and vit.

Prevention & Control

1. Sanitary disposal of Human excreta.
2. Mass Treatment
3. wearing shoes and gloves
4. Killing filariform larva using soil larvicides
5. Health education.

(Ground itch)

* occurs due to Penetration of skin by filariform larva of *Anchytostoma duodenal*.

* This Result in.

Dermatitis

itching

burning

followed by

Erythema

Edema.

* Soon Papule formation and eruption of vesicle followed by 2nd bact. infection.

* disappear 1-2 weeks.

hair cylindrical snake like.
* Trichostrongylus axei *
الموضوع

Disease: Trichostrongyliasis.

* Distribution: Cosmopolitan, especially agriculture area.

Habitat: upper part of small intestine of herbivorous animal and occasionally man.

Definitive host

Diagnostic stage: egg in stool

Infective stage: Filariiform larva

Mode of infection: ingestion of contaminated food or drink containing Filariiform larva

Life cycle:

Adult in small intestine → eggs → feces → soil
Rhabditiform larva → Molt 2 times
→ Filariiform larva → ingestion → Small intestine
3rd molt → Penetration villi → back to lumen
→ Adult

Pathogenesis

light infection → no symptoms

Heavy infection → anemia

Sign of cholecystitis

Diagnosis:

* Finding egg in stool

* duodenal aspirate

* Stool culture 1 day gave larva

Treatment

Thiabendazole (Mintezol)

cylindrical like. stool

Strongyloides stercoralis

Disease: strongyloidiasis.

Distribution: Cosmopolitan more in tropical and subtropical countries.

Habitat: Duodenum and upper jejunum

Definitive host: Man.

Diagnostic stage: Filariform larva. ***

Infective stage: Filariform larva. ***

Mode of infection: Penetration of skin or mucosa of intestine.

Life cycle: Adult in small intestine → eggs inside

The mucosa of intestinal villi → Rhabditiform

Life cycle: larva → lumen → Faeces

→ 3 Type of life cycle.

Direct
Indirect
Auto infection

Direct life cycle:-

Rhabditiform larva → soil → moult → infective

Filariform larva → Penetrate skin → venous circulation

→ lung → alveoli → Penetrate wall of alveoli → migrate

Through trachea → Swallowed → small intestine.

→ 2 moult → Adult.

Indirect life cycle (if soil condition is optimal)

Rhabditiform larva → soil → 4 moult within 2 days

→ adult → mature ova → Rhabditiform larva

(Free living) as long as the condition are suitable.

If the condition become unsuitable. The Rhabditiform larva become infective Filariform larva.

Auto infection.

When the person suffer from constipation, Rhabditiform larva

have enough time to moult into infective Filariform larva.

Then they Penetrate mucosa of large intestine. Then.

Complete the cycle. (internal auto infection)

Pathogenesis and clinical picture

Skin lesion:-

- also ~~for~~ infective Flariform larva. Can Penetrate Peri anal skin. after coming out from anus and then. Complete the cycle (external auto infection)

Pathogenesis and clinical picture..

Skin lesion (Cutaneous)

- Dermatitis, itching at site of Penetration.
 - Cutaneous larva migration. larva currens
- [lesion start at Peri anal Region Result from external auto infection. The larva Remain in The skin and extend as linear eruption across The buttock, thigh, back

Migration stage (lung lesion)

Pass of The larva to lung lead to minute hemorrhage.
Pneumonitis, Fever, Cough, Hypostis, Eosinophilia.

intestinal stage

- Peptic ulcer & Malabsorption syndrome.
- Steatorrhea, Mucous diarrhea, ulceration, necrosis.
- secondary bacterial infection.

Disseminated Strongyloidiasis $\leftarrow$ heavy infection. impaired immunity.

Some larva Pass Through The Pulmonary barrier To left side of Heart to Reach to Various organ of body

also in Patient with impaired immunity The Parasite Produce massive number of larvae which Penetrat Extra intestinal organ. and becom fatal it called opportunistic Parasite.

Diagnosis

- 1] clinical sign.
- 2] Rabditi Form. larva in stool and give adult in culture
- 3] Esino Philia
- 4] Examination of sputum for larva.
- 5] Serology → ELISA & IFAT

Treatment

- (1) Thiabendazole.
- (2) Albendazole.
- (3) Ivermectin.

Prevention and Control ✓

Capillaria Philippinensis.

Disease ~~is~~ intestinal capillariasis.

Distribution. Philippines, Thailand, some cases in Egypt

Habitat. ~~in~~ fish. eating birds and occasionally man.
embedded in mucosa of jejunum, ileum.

Definitive Host

Diagnostic stage. Egg in stool.

Infective stage infective larva in fish intestine.

Mode of infection. Through eating raw, poorly
cooked fish and internal auto infection

Life cycle

Adult female $\rightarrow$ egg $\rightarrow$ Fresh water.
 $\rightarrow$ embryonated egg $\rightarrow$ intermediate host (Fish)
 $\rightarrow$ Small intestine of fish $\rightarrow$ Larvae $\rightarrow$
 $\rightarrow$ ingestion of fish by Man $\rightarrow$ Small intestine
of man $\rightarrow$ Adult

*** Auto infection:-

Female $\rightarrow$ larvae $\rightarrow$ invade mucosa $\rightarrow$ Adult

Pathogenesis.

- infection lead to chronic inflammatory reaction.

$\rightarrow$ Atrophy of villi $\rightarrow$ Malabsorption of
fat, sugar, Pn., electrolytes

Clinical Picture

- Abdominal Pain, colic, chronic diarrhea.

- dehydration, loss of weight, low grade fever

- Edema in lower limb (hypoproteinemia)

Diagnosis 1- clinical manifestation.

2- Egg in stool and larva in heavy infection.

3- Hypo Proteinemia.

4- low blood Calcium, Potassium, and cholesterol

Treatment

Specific Treatment

Mebendazole.

Albendazole.

Supportive Treatment

- High ptn. diet.

- fluid and Electrolyte Replacement

Cutaneous larva migrans

Condition. Result from invasion of skin of man by filariform larva of dog and cat hook worm. (*A. Caninum* & *A. braziliense*.) larva fails to creep beyond the germinal layer of skin but migrate from place to place.

Pathogenesis and clinical picture:-

1. lesion starts as red itchy papule at the site of entry followed by slightly elevated erythematous serpiginous tunnel with itching and secondary infection.
2. lesion advances at the rate of 1-2 cm/day for several weeks or months till larva die.

Diagnosis.

- Clinical :-
- advancing & serpiginous tunnel.
 - History of contact of skin with soil.
 - larva always seen in head of tract.
 - high Eosinophilia.

Treatment:

1. Albendazole.
2. Thiabendazole ointment.
3. Thiabendazole (mintezol).
4. Antibiotic for 2nd infection.
5. Antihistaminic.

Other causes of cutaneous larva migrans?

1. Human and non human strains of *Strongyloides* (larva currents).
2. Cutaneous myiasis caused by larva of flies as *Gastrophilus* and *Hypoderma*.

Visceral larva migrans

Condition. Result from invasion of human visceral organs by larva of dog and cat ascaris.

Toxocara Canis ← الأسكاريس الكلب

Toxocara cati ← الأسكاريس القط

Life cycle:

Adult ~~larva~~ live in small intestine of dog and cat → man is infected by ingestion of Toxocara canis or cati which passed with the stool of cat or dog. → larva hatch → Penetrate intestinal mucosa and carried by blood to liver, lung, eye and other organ and causing inflammation.

Pathogenesis

Presence of larvae in viscera stimulate formation of Eosinophilic granulomatous lesion.

Clinical Picture

Depend on location of larvae

1. Hyper Eosinophilia
2. Enlarged Tender liver
3. Pneumonitis and cough
4. ocular toxocariasis (Retinochoroiditis, Retinitis.)
5. Neurological disturbance asepilepsy.

Diagnosis

1. Marked eosinophilia and hyper gamma globulinaemia
2. serology :- EIISA by using toxocara larva antigen.
3. Laparoscopy and biopsy of liver nodules

Other Causes

Heavy infection of Ascaris, Ancylostoma Strongyloids.

Treatment

1. Albendazole

2. Corticosteroid to decrease Allergic Condition.

Larva migration

Migration of larvae of nematode in unsuitable host larvae can't complete its cycle and cannot reach to Adult stage.

Trichinella spiralis

Disease: Trichinosis

Distribution: cosmopolitan especially in Pork eating countries.

Habitat: Small intestine.

Definitive host: Small intestine of man, pigs and Rodents (Rats)

Diagnostic host:

Intermediate host: man, pigs, Rodent.

Infective stage: Encysted larvae

Mode of infection: ingestion of improperly cooked infected Pork.

Life cycle:

larvae → ingestion by Pigs → its muscle
→ ingestion by man → Small intestine.
→ 3 moults → adult → Female →
eggs in mucosa → larva → blood →
all Tissue especially striated muscle. → encyst
→ Encysted larvae.

Pathogenesis and clinical picture.

intestinal stage (1st week)

* gastro-enteritis → nausea, vomiting, abdominal cramps
diarrhea (similar to food poisoning.)

Stage of migration (2nd week)

- Fever ~~Recover slowly~~ - eosinophilia.
- Muscle Pain. - edema of eyelid
↳ myositis, weakness of invaded muscle.

Encapsulation stage (3rd week)

- * Fever Recover slowly
- * Muscle Pain Persistent
- * Pneumonia, encephalitis in severe infection

Diagnosis:-

Clinical:-

History of eating Pork with Fever, Esino Philia
fascial edema, Myositis.

Laboratory:-

- Muscle biopsy → Examined for larvae.
- Esino Philia
- Intra dermal test. [Bachman test]
- Serological test as IFAT & ELISA
- X-Ray showing calcified cyst.

Treatment

1. Mebendazole or Thiabendazole
2. Corticosteroid
3. Symptomatic treatment for fever, headache,
Muscle Pain.

Prevention & control

1- destruction of Rats and Proper breeding Pigs

2- Heat Treatment of ~~gar~~ garbage

3- Meat inspection of Slaughter house.

4- Proper cooking of Pork

	<i>Ascaris lumbricoid</i>	<i>Trichuris Trichiura</i>	<i>Enterobius vermicularis</i>	<i>Ancylostoma duodenale</i>	<i>Trichostrongylus Colubri formis</i>	<i>Strongyloides stercoralis</i>	<i>Capillaria philippinensis</i>	<i>Trichinella spiralis</i>
Habitat	Lumen of small intestine	Caecum and adjacent part	Caecum and adjacent part	Small intestine (Jejunum)	Upper part of small intestine of herbivorous animal and occasionally man	Duodenum and upper Jejunum	Fish eating bird and occasionally man embedded in mucosa of Jejunum, ileum.	small intestine
Disease	Ascariasis	Trichuriasis	Enterobiasis	Ancylostomiasis	Trichostrongyliasis	Strongyloidiasis	Capillariasis	Trichinosis
Distribution	Cosmopolitan	Cosmopolitan	Cosmopolitan	Mediterranean. North Africa, South America, China, India	Cosmopolitan. especially agriculture area.	Cosmopolitan in Tropical and Subtropical countries.	Philippines, Thailand. Some cases in Egypt.	Cosmopolitan. especially in pork eating countries.
Def. H	Man.	Man.	Man only.	Man only.	herbivorous animal.	Man.	Fish eating bird.	small intestine of Man, Pigs, Rodents.
I.S	Ascaris egg in stool	Egg in stool	egg found mainly in Peri anal Sxk (become infective in few hours)	Egg in stool.	egg in stool	Flariform larva	Egg in stool.	
Mode of infection	embryonated egg	Embryonated egg.	infective egg containing larva.	Flariform larva	Flariform larva.	Filariform larva.	infective larva. in fish intestine.	Encysted larva.
	Ingestion of an embryonated egg in contaminated food or drink.		ingestion of infective egg containing larva through. (1) Auto infection. (2) Contaminated food or drink. (3) Air born infection. (4) Retro infection.	Penetration of flariform larva skin of Man when he walk bare foot on soil containing larva.	ingestion of contaminated food or drink containing flariform larva.	Penetration of skin or mucosa of intestine.	eating raw, poorly cooked fish and internal auto infection	ingestion of improperly cooked infected pork.
				Ground H.A.	anemia sign of cholecystitis.			

infective larva
Comma shaped larva

Tissue Nematodes

- * Adult nematode live in tissue of man.
- * female lays larvae no eggs (larviparous)
- * Transmission of this nematode need intermediate host called Arthropod Vector (الكشرات)
infection. ← ينقل الـ vector

Vector → an arthropod that take the larva from diseased human to develop inside its body before transmitted to healthy human.

longest nematode. ← Dracunculus medinensis (التيين) (Dragon worm & Guinea worm) Medina worm

Disease. Dracunculiasis

Distribution. in area where people depend in wells as ^{الآبار} Habitat. Source of water (Sudan, Mali, Ghana)

Habitat. sub cutaneous tissue.

Definitive host infected human

Reservoir host Dog, horse, cattle.

Diagnostic stage. Coma-shaped larva

Intermediate host Cyclop.

Infective stage. infective larva

Mode of infection. Drinking water containing infected cyclope.

Life cycle

Adult → copulation → male die → female.
migrate to subcutaneous tissue. especially that become connected with water → during contact with water → uterus prolapses → discharge larvae until they finished → ingestion by cyclope → body cavity → moult → ingestion of cyclope by D.H or R.H

التاريخ الموضوع
Larva migrate Through wall of small intestine.

→ Retro Peritoneal tissue → maturation.

Pathogenesis and clinical Picture.

Allergic Reaction -

Allergic Reaction Due to metabolic product of worm → urticaria, Rash, nausea, vomiting, diarrhea, asthma.

Skin blister

2. skin opposite anterior end show Red Papule Then blister which ulcerate.

2nd.

3. 2nd infection of ulcer lead to abscess, Cellulitis and septicemia.

Forced

4. forced extraction of female → severe allergic Reaction

Clinical

Diagnosis

1. outline of worm under skin may be seen
2. skin lesion (Papule, blister, ulcer)

Laboratory

- obtaining The larvae by placing the limb in water
- Eosinophilia
- intradermal test
- X-Ray show calcified female.

Treatment

(I) Removal of The worm.

- Rolling The worm on a stick → pulling gradually each day ~~until~~ to avoid Rupture of worm.
- Surgical Removal

[2] Thiabendazole to Reduce inflammation around the worm.

[3] Anti septic dressing

[4] Anti biotic

[5] Anti histaminic

Control

- 1- Eradication of cyclops wells (سحب الماء في الآبار بمقومات cyclops)
- 2- Boiling or filtering well's water
- 3- use Pumps

3 intestinal nematode.

<i>S. stercoralis</i>	<i>C. Philippinensis</i>	<i>Trichinella spiralis</i>
الموسى Penetration Female وكل ال eggs وال eggs يتلف موسى ال larva وهو ال موسى ال egg تسر وتخرج ال larva وتخرج ال موسى وتنتشر ال lumen وتنتشر ال stool	الموسى Penetration Female وقية نوسم ال Female نوع ال eggs ونوع ال larva تنتشر ال stool الموسى ال adult	الموسى Penetration Female larva وكل mucosa وال larva تسر وتنتشر Blood stream وتنتشر Encysted larva وتنتشر in muscle of Patient
⇒ Inflammatory Reaction in mucosa	inflammatory Reaction of mucosa + Atrophy of villi	inflammatory Reaction of mucosa
⇒ Diarrhea alternate with constipation.	Abdominal Pain, vomiting. diarrhea, dehydration.	Abdominal cramp nausea, vomiting, diarrhea
⇒ Burning epigastric Pain Tenderness	Electrolyte imbalance. mal-absorption. edema in lower limb	Similar to food Poisoning.

Filaria (Thread worm)

Thread like - cylindrical worm.

Filaria worm can affect human.

Lymphatic
System.

Subcutaneous
Tissue.

Serous
Cavities

① *Wuchereria bancrofti*

① *Loa Loa*

① *Mansonella Perstans*

② *Brugia malayi*

② *Anchocera Volvulus*

② *Mazzardi*

⇒ Filaria Transmitted to man Through bite
of Arthrocod (mis)

Wuchereria bancrofti

Disease bancroftian filariasis, Elephantiasis

Distribution in tropical and sub tropical area in Africa, Asia, South America.

Found in Egypt in

○ Kaiohia

○ Dakahlia

○ Shar Kia

○ Gizza, Assiut.

Habitats: lymph vessels and lymph gland

Definitive host: Man only.

Diagnostic stage: Microfilariae but adult may be seen.

Intermediate host Mosquito

Infective stage: infective filari form larvae when mosquito bites the man

Mode of infection: when mosquito carry infective filari form larva bites the man.

Life cycle:

Adult → Lymph. gland of Man → Microfilaria
→ lymph vessels → blood (appear in peripheral blood at night (nocturnal periodicity) → Mosquito during biting and sucking of infective host → development inside mosquito → infective filari form larvae → go to man again during biting. → enter by Penetration or through wound or any abrasion → Pass to lymph node and vessels → Maturation → adult

„ cyclo developmental Transmission “

Parasite. Develops in body of mosquito with out increase in number.

Pathogenesis and clinical Picture

[1] Asymptomatic

Many infections are asymptomatic especially in Endemic area. Diagnosis only by blood Examination.

[2] Acute inflammatory Phases

Due to immunological Reaction to toxic Product of worm & 2nd infection by Streptococci

- symptom appear about one year after infective bite.
- ★ attack of lymphangitis → vessels appear raised, hot, Red swollen, Tender Commonly in limb especially in legs & genitalia (Funiculitis, orchitis, Epididymitis, epididymo-orchitis)
- ★ attack of lymphadenitis → Enlarged and tender lymph node
- ★ Filarial or elephantoid Fever
 - sudden onset with Rigor
 - sweating
 - lasts for Few hours or several days
- ★ Bacterial & fungal super infection.

[3] chronic Phase :-

- ★ obstruction of lymphatic → Due to fibrosis of lymph vessels and node following Repeated attack of lymphadenitis and lymphangitis and also due to coiled worm in inside lymphatic
- ★ Distention of lymph vessels → lymphedema and varicosis.
- ★ Rupture of distended lymphatic
 - In The scrotal sac → Hydrocoele.
 - In The Pleura → chylo-Thorax.
 - In Peritoneal cavity → chylous ascites.
 - In urinary tract → chyluria.
 - In intestine → chylous-diarrhea.

★

* Lymph edema → increases permeability of lymph vessels wall
lead to leakage lymph into Tissue

* Elephantiasis:

↑ Permeability of the wall of obstructed lymphatic →
leakage of lymph with high concentration of Protein under
skin → deposition of fibrous tissue → the skin and
underlying tissue become hard, dense, non pitting. (hard edema)
→ the skin appear thickened, rough, fissured.
Susceptible to 2nd infection → huge
enlargement of affected part usually, Legs, scrotum,
Vulva, breast, arm.

→ clinical

① Diagnosis

① clinical sign and symptoms.

② Laboratory:

* ^{by finding} Recovery of microfilaria in blood. Through.

(a) → Examination of drop of fresh blood show movement
of microfilaria.

(b) → Di-ethyl-carbamazine Provocative test :-
giving 100 mL of (DEC) → Taking blood 45 minute
later → Microfilaria can demonstrated at any time
of day.

(c) → Concentration method (Knot's technique) :-
10 mL of blood + equal volume of 2% formalin in
centrifuge tube Then centrifuge → sediment
is separated then spread on slide and stained with
Giemsa stain and examined for microfilaria.

(d) → Eosinophilia.

⑤ — Immuno diagnosis

- * intra dermal test
- * serological test ELISA
- * Detection of circulating antigen in blood.

⑥ — Examination of chylous fluid and fluid aspirated from hydro coele and Enlarged lymph.

⑦ — Detection of adult in lymph node biopsy.

[3] Radiology. "imaging techniques"

- * X-Ray show calcified worm.
- * ultrasonography show moving activity of adult worm

Treatment:-

① DEC — Kill adult and modifies microfilaria to be easily attack by immune system.

② Ivermectine — Its effect Remove microfilaria from blood but not affect on adult worm.

③ Combine of DEC & Ivermectine — give better Result

④ Symptomatic treatment:-

anti biotic and anti fungal to Prevent 2nd infection
Anti histaminic to Prevent allergic Reaction.

⑤ Surgical Treatment in

- * chronic hydro coele
- * elephantiasis.

Supra l. 12

occult Filariasis

Tropical Pulmonary Eosinophilia

Adult worm and their microfilaria are found in tissues of patient (not found in lymph and blood vessels)
→ Present in Liver, Lung, Spleen.
* No microfilaria are found in blood.

⇒ when adult and their microfilaria are found in Lung tissue we call this condition TPE
Tropical Pulmonary Eosinophilia.



Type of occult filariasis characterized by

- hyper Eosinophilia.
- lymph node Enlargement
- Pulmonary symptoms (dry nocturnal cough, dyspnea, asthma, wheezing)
- Low grade fever

Due to Allergic Reaction To microfilaria in lung.

Diagnosis:-

- (1) No microfilaria found in blood.
- (2) serological Test with filarial antigens usually strongly Positive.
- (3) High Eosinophilia.
- (4) X-Ray show mottled shadow

Treatment:-

- (1) D.E.C

Loa Loa.

Distribution:- Common in ^{المناطق} Tropical Africa

Diseases:- eye worm, Loiasis, Calabar swelling.

Habitate:- Sub cutaneous Tissue.

Infective stages:- infective larva

Diagnostic stage:- Microfilaria (in day). Urinary.

Pathogenesis:-

① Calabar swelling (fugitive).
sub cutaneous.

Painful, non-pitting swelling. Come suddenly and disappear gradually. and occur in any part of body.

This swelling due to allergic reaction to parasite and its product.

② Adult worm pass in front of eye ball under conjunctiva.

Diagnosis:-

① clinical

② laboratory.

→ Finding Microfilaria in blood in day.

→ Eosinophilia

→ immunodiagnosis.

→ P.C.R.

Treatment.

* DEC

* Surgical Removal.

* Anti Histaminic.

(Onchocerca volvulus)

Distribution:- Tropical ^{Africa} and central America.

Vector:- Simulium (black fly).

Disease:- river blindness / onchocercosis.

Habitat:- sub cutaneous tissue in bony area.

Infective stage:- infective larva in saliva of simulium.

Diagnostic stage:- Microfilaria in skin

Pathogenesis.

① onchocercoma:-

* Painless, firm, movable subcutaneous nodules which vary in size from few mm to about 10 cm.

Contain the parasite.

* Present over bony prominence.

* nodule causes no medical problem unless present near to vital organ.

* This lesion occurs due to inflammatory reaction to dead or living parasite.

② ocular lesion (River or Sudan blindness).

Caused by migration of Microfilaria from scalp tumor to eye. → Toxic and allergic reaction to living and dead microfilaria.

- (1) Keratitis (inflammation of cornea)
- (2) iridocyclitis (inflammation of iris)
- (3) Retinitis (" " Retina)
- (4) Chorioiditis (" " choroid)
- (5) optic neuritis (" " optic nerve)

(3) Skin lesion.

Caused by Migration of Microfilaria in skin.

- (1) Hanging groin. $\Rightarrow$ occur in hip region in which sac formed and may contain enlarged lymph node
- (2) depigmentation and hyper pigmentation. (sowda)
- (3) Dermatitis.

Diagnosis:-

- (1) Clinical
- (2) Laboratory

* Determination of Microfilaria in skin.

* Mazzotti test

oral administration of single dose of 50 mg D.E.C.
 $\rightarrow$ Reaction to the test start 2-24 hour later.
Due to death of Microfilaria with intense Pruritus, and Rash, Fever

(3) Patch test

D.E.C. applied locally on the skin of patient.

(4) Seriological test.



80
11

Loefflers syndrome:-

Condition occurs due to migration of larva of nematode to the lung as (Ascaris lumbricoides - Ancylostoma duodenale and Strongyloides stercoralis).

Migration of larva to lung leads to Pneumonitis with Cough, wheezing, Low grade fever and Eosinophilia.

X-Ray shows scattered mottling appearance known as Loefflers syndrome.

[2] Complication of Ascaris Lumbricoides ?? ✓

[3] Visceral Larva Migration ??

Parasitic infection caused by invasion of human tissue by larva of Toxocara canis, Toxocara cati also occur in heavy infection by larva of

- * Ascaris Lumbricoides.
- * Ancylostoma duodenale.
- * Strongyloides stercoralis.

when the larva escape the lung to left side of heart and distributed with systemic circulation.

To different organs causing formation of Eosinophilic granulomatous lesion.

[4] Ground itch.

occurs due to penetration of skin by filariform larvae of Ancylostoma duodenale and lead to

Dermatitis, itching, burning. followed by erythema and soon papule formation and eruption of vesicle. followed by secondary bacterial infection.

It disappears 1-2 weeks.

Hyper infection syndrome:-

- * un Controlled infection by *S. stercoralis*.
- * it occurs in immunosuppressed patient.

→ in Pulmonary hyper infection:-

Loeffler syndrome with coughing, wheezing, asthma and haemorrhage.

also some larva pass through pulmonary barrier and reach to left side of heart pass with systemic circulation to different organ.

→ in intestinal hyper infection:-

symptoms include diarrhea, nausea, vomiting, abdominal pain, Loss of weight.

↳ Bacteremia is common complication of hyper infection syndrome. Caused by larva carrying bacteria to the blood stream.

The mortality rate with bacterial infection is high. Reach to 90%.

Disseminated strongyloidiasis.

wide spread of larva migration to all organ and tissues of the body as Liver, heart, L.N, Gall bladder, Kidney, Pancreas, brain.

it occurs in cases of immunosuppressed patient and hyper infection.

Complication.

- 1- cholecystitis
- 2- Pancreatitis.
- 3- Peritonitis.
- 4- Paralytic ileus
- 5- Petechiae and Hmg.
Due to larva migration
Through wall of blood vessels
- 6- Brain (headch, meningitis, coma)
- 7 infarction.
- 8- intestinal Perforation.
- 9- sepsis.

D.E.C Diethyl Carbamazine Provocative test :-

- This test used in diagnosis of w. bancrofti infection in area where Microfilaria have nocturnal Periodicity.
- it induce Microfilaria to appear in Prepheral blood. Through Treatment by single dose 100mg of D.E.C. blood sample Taken 45 min. after administration of D.E.C then Microfilaria can detected in blood.

occult filariasis?

Adult worm and their Microfilaria are found in tissue of Patient don't reach to Prepheral blood because they are destroyed in tissue.

in this cases the Patient develops massive Eosinophilia, Generalized Lymph node Enlargement, hepatosplenomegally and Pulmonary symptoms.

Tropical Pulmonary Eosinophilia ?? T.P.E

Type of occult filariasis characterized by.

- (1) hyper Eosinophilia.
- (2) loss of weight.
- (3) Lymph node Enlargement.
- (4) Pulmonary symptoms as Dry Cough, dyspnea, asthma, wheezing.

Diagnosis.

- (1) No Microfilaria found in blood.
- (2) High Eosinophilia.
- (3) Serological test with filarial antigen usually strong.
- (4) Marked increase in total serum IgE
- (5) X-Ray show mottled shadow.

Treatment.

D.E.C.

Calabar Swelling :-

- * The most common pathology associated with Loa Loa infection.
- * Result from Allergic Reaction to Parasite and its Product.
- * This swelling characterized by.
 - (1) Painful non Pitting Swelling. "sub cutaneous"
 - (2) Come suddenly and disappear gradually. and Lasts usually from 1-3 day and accompany by. urticaria, and Pruritis.
 - (3) Found any where in The body. but most common. in extremities. especially back of hand and arm. near to Large joint.

onchocercoma :- ??

→ Lesion develop in onchocerca volvulus infection.

- * sub cutaneous nodule painless, firm, movable. oval in shape. , single or matted together.
- * Vary in size from few mm. to about 10 cm.
- * it Present against bony Prominence. such. as knee. , hip, iliac crest, Sacrum, Ribs, Scapula, elbow, scalp.
- * Scalp nodule May invade The bone of skull.
→ Necrosis of bone. due to Mechanical Pressure. and infiltration. The brain tissue.
- * This lesion occurs due to inflammatory. reaction To dead or living Parasite.

Sowda ??

Lesion develop in onchocerca volvulus infection characterized by.

* Hypertrophy of skin with either hyper pigmentation or de pigmentation.

* it occurs in white people in Yemen due to hyper sensitivity reaction to toxic product of dead Microfilaria.

* Sawda manifested Clinically by localized form of onchocerciasis mainly to one limb but may involve arm or trunk.

* it characterized by.

✓ (1) intense itching.

✓ (2) involved skin become swollen, dark with papule.

✓ (3) Enlargement of drainage lymph node.

River blindness (sudan blindness)

* Ocular Lesion Related to intensity of Microfilaria of onchocerca Volvulus in eye structures.

* ocular lesion Result of inflammatory Reaction to The Trapped Microfilaria in different eye structure.

* ocular lesion include.

(1) Keratitis (inflammation of Cornea)

(2) iridocyclitis (" " iris)

(3) Retinitis (" " Retina)

(4) Conjunctivitis Causing irritation and Lacrimation.

(5) choroiditis

(6) Optic neuritis

(7) Photo Phobia.

Mazzotti test :-

Test used for diagnosis of onchocerca volvulus infection.
→ oral administration of single dose 50mg of DEC
Resulting in appearance of Mazzotti Reaction.
which start 2-24 hours later due to death of
Microfilaria. This reaction include.
intense Pruritis, Rash, fever.

Elephantiasis :-

- * late complication of w. bancrofti infection,
- * Resulting from repeated lymphadenitis and lymphangitis which result in.
 - 1- obstruction of lymphatic due to fibrosis.
 - 2- Distention of lymph vessels
 - 3- lymph edema.

This followed by.

- increase permeability of lymph vessels → leakage of lymph to tissues with high concentration of protein under skin. → fibrous tissue deposition → skin change (thickened, rough, fissured, non pitting edema, dense)
- 2nd bacterial infection.
- huge enlargement of affected part (leg, vulva, scrotum, breast, arm).

Diagnosis of w. bancrofti

1] clinical sign and symptoms.

2] laboratory:

- examination of fresh blood show microfilaria movement
- D-E-C Test:
 - ✓ giving the patient 50 mL of DEC
 - ✓ Taking blood 45 min. later.
 - ✓ Microfilaria can be demonstrated any time of day.
- Concentration Method (Knott's ~~technique~~ technique).
 - 10 mL of blood + equal volume of 2% formaline.
 - Centrifugation
 - Separate sediment then spread on slide and stained with Giemsa stain and examined microscopically.
- Immuno diagnosis by
 - ✓ intra dermal test
 - ✓ Serology by ELISA
- Examination of chylous fluid.
- Esino Philia.
- biopsy and detection of Adult.